

P26**Lifestyle, IGF-I and prostate cancer**

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In previous studies we reported that a low-fat, high-fiber diet and exercise program altered serum factors that resulted in reduced growth and increased apoptosis of serum-stimulated, androgen-dependent LNCaP and LAPC-4 cell lines. In the present study we investigated the role of IGF-I in mediating these responses by comparing pre and post intervention serum stimulation of LNCaP and PC-3 (androgen independent cells). Growth was assessed by the CellTiter 96AQ, MTS assay and apoptosis by the Cell Death Detection ELISA Plus assay. A low-fat, high-fiber diet and daily exercise intervention reduced growth of LNCaP cells by 43.6% and increased apoptosis by 354%. When the IGF-I receptor blocker aIR3 was added to the pre- and post-intervention serum, growth of LNCaP cells was reduced to the same level (~41% of pre) and apoptosis increased to the same level (~425% of pre). When IGF-I (60 ng/mL) was added back to the post-intervention serum, the reduction in growth and increase in apoptosis were eliminated. In androgen independent PC-3 cells, diet and exercise intervention only reduced growth by 26.4%. When aIR3 was added to the pre samples, growth was reduced by only 32.8%. Diet and exercise increased apoptosis in the PC-3 cells by only 31.8% and when aIR3 was added to the pre samples apoptosis was increased by only 214.6%. When the PI3 Kinase blocker (LY294002) or the p38 MAP Kinase blocker (SB202190) were added to the pre serum-stimulated LNCaP cells there was an additional 10% reduction in growth compared to the aIR3 results. With the two blockers apoptosis was increased by an additional 299.6% (LY294002) and 308.3% (SB202190) compared to the aIR3 results. These data indicate that in early stage, androgen dependent PCa, diet and exercise intervention significantly reduces growth and induces apoptosis of LNCaP cells primarily by reducing serum IGF-I. In addition, some other serum factor(s), not responsive to diet and exercise, stimulate growth and especially suppress apoptosis in the LNCaP cells. Diet and exercise is far less effective in reducing growth and inducing apoptosis in androgen independent PC-3 cells.

P27**Role of positive family history in the development of gastric cancer: a meta-analysis study**

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Background: The role of family history in gastric cancer (GC) has been showed by several studies. In this study we aim at doing a meta-analysis of case-control studies evaluating the rate of positive family history of GC in patients versus controls.

Methods: A comprehensive English literature search was performed using PubMed from 1966 to September 2005 based on the keywords: Gastric, stomach, cancer, family history, and

risk factor. 120 potential articles were reviewed, and review articles on relevant topic were complementarily scrutinized. Only case-control trials comparing the rate of positive family history in patients with gastric cancer versus non-cancer controls were included. All articles retrieved were appraised critically. Meta-analysis of pooled odd ratio (fixed model) was performed by Review Manager 4.2.

Results: Nine case control studies were included (3 from Europe, 3 from Japan, and one from each of North America, Turkey and Taiwan). One study was excluded after analysis as it had an out of range result (OR: 10.3) so the meta-analysis included 8 studies with 2494 patients in GC group and 6628 in control group. 27% of the patients and 15% of the controls had at least one relative with GC ($P < 0.0001$). Odd Ratio to have a positive family history for patients with GC to the controls was 1.99 (95% CI: 1.82-2.17) (figure 1). By considering positive history only in the first-degree relatives 28.2% of the patients and 13.8% of the controls had at least one relative with GC ($P = 0.0062$). Odd Ratio to have a positive family history for patients with GC to the controls was 2.16 (95% CI: 1.93-2.42).

Conclusions: It seems that patients with gastric cancer have a double chance of having at least a case of GC in family. This is even more if this positive family history is in the first-degree relatives.

P28**Seasonal occurrence of spontaneous mammary tumors at mice C3H/He**

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A well-studied model of neoplastic transformation and progression is the breast cancer system of the C3H mouse. Spontaneous and transplanted tumors are described. Nevertheless the moment of occurrence of spontaneous tumors is not adequately studied. The major focus is on a experimental approaches to the study of mechanisms and crucial moment of a rise of spontaneous mammary tumors of C3H/He mice. Change of a level of estrogens receptors is studied also. The results obtained to date suggest that tumor cells may originate in mice shortly after pairing. This moment is calculated from experimental data. Both the originating of tumors and a breeding equally depends on seasons (daylight hours). Obtained data confirm a greater role of steroid hormones for initialization of formation of tumors. Probably hormonal influence is a final stage of a beginning tumorigenesis. Steroid hormones do not act as carcinogens. They play a role only as regulators of a transcription. Hormones change a state of cells. Where the formalized state of a cell is history their gene transcription and a pattern gene transcription at present. Experience Bitner is successfully repeated. Synthetical steroid hormones were as the "factor of milk". No histological feature has been found which might explain the variation in levels of estrogen receptors in mice mammary tumors. Growth of transplanted ER-positive mammary tumors was modulated by oestrus cycle of mice. Emphasis is given to common mechanisms of action steroids in mice or/and human as a guide to therapy and prevention in men. Ability of tumoral cells to adapt for change of a surrounding microenvironment answers on a question on a paradoxicality of a hormone therapy. Series of parameters of a tumor such as the invasiveness,

heterogeneity and others, are consequence of adaptive properties of tumoral cells. The description and evaluation of concepts and the future projections in cancer research will be interest to all investigators concerned with cancer endocrine tissues.

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Communication and educational model for cancer prevention in Colombia

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Introduction: Colombian National Social Security includes Health Education as a strategy for Health Promotion and Preventing Disease. Cancer prevention is focused on Information, Education and Communication strategies to control cancer risks factors by promoting behavioural change. Evaluation of these strategies failed to live up to public and professional expectations.

Objectives: To design an Education Model for Cancer Control within a Cancer Control Model in order to improve control of cancer risk factors, promote cancer early detection in Colombia.

Methodology: a non systematic review of literature was done using the following words: health education, education for cancer control and Behavioural Models. analyzing the literature, an Educative-Communication Model was proposed. This proposal was discussed with experts from different regions around the country.

Results: We defined a Model grounded in the rational structure of Third Generation Models for health education. It is dialectical model on the bases of social interaction. In order to design communication products it is necessary to gather needs socio anthropological information, communitarian "habitus" and risk perceptions. Communication products are essential elements for establishing a dialogue between communities and institutions. The proposal should consider the cultural and political framework in order to modify it, if necessary.

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The clinical-genetic monitoring of cancer of female reproductive organs as a problem of preventive medicine

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The specialized genetic consulting of relatives of patients with malignant tumors of female reproductive organs doesn't function in Ukraine. The decrease of mortality and incapacity of patients with this kind of cancer is impossible without the state program of anticancer actions.

Goals of the study: The assessment of the efficacy of the genetic approaches to the early diagnostics and prevention of tumors of female reproductive sphere (patients with ovarian and endometrial cancer), the creation of groups of increased genetic

risk among their relatives and clinical-genetic monitoring of their health on the example of Chernivtsi Region of Ukraine. Materials and methods. The clinical-genealogical analysis has been conducted among 500 women with ovarian cancer and 482 women with endometrial cancer.

Results and discussion: The clinical-genealogical analysis revealed a high level of aggregation of malignant tumors among relatives of patients. 110 healthy women have been picked out in the genealogies of patients with ovarian and endometrial cancer, who were included in the group of genetic dispensary follow-up. 62 women were close relatives of the patients with ovarian cancer (30 – sisters, 22 – daughters, 10 – mothers) and 48 women were relatives of the patients with endometrial cancer (24 – sisters, 17 – daughters, 7 – mothers). All women from the risk groups underwent detailed screening examination. During the observation period of 3 years on 110 women from the risk groups several cases of benign tumors, pre-cancer of the female reproductive sphere have been diagnosed: myoma of the uterus – 8 patients; ovarian cysts and cystomata – 7 patients; nodular and diffuse mastopathy – 29 patients; tuboovarian tumors – 5 patients; endometrial hyperplasia – 9 patients; chronic adnexitis – 11 patients. This approach in the decision of the problem of early diagnostics of cancer of reproductive sphere is very efficient – four women from the group of enhanced genetic risk have been first diagnosed with early ovarian and endometrial cancer.

Conclusions: The information of the clinical-genetic monitoring of the health of persons with enhanced risk of ovarian, endometrial and breast cancer, confirms the high efficiency of this approach in the decision of problems of early diagnostics and prevention of malignant tumors of female reproductive system. It should be recommended as a model for creation of a new direction in the anticarcinogenic fight.

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Different pathways of centromeric micronuclei formation in peripheral blood lymphocytes of cancer patients and workers occupationally exposed to mutagens/carcinogens?

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Aim: The cytokinesis blocked micronucleus (CBMN) assay is widely used in biomonitoring purposes. Micronuclei (MN) are either a recognized consequence of genome instability or thought to be a biomarker of exposure to environmental mutagens/carcinogens. To discriminate between MN containing acentric chromosomal fragment and MN containing whole chromosome, in situ hybridization can be performed. MN are then commonly classified as centromeric or acentromeric but one can also discriminate between monocentromeric and multicentromeric MN. We report data from 3 biomonitoring studies in which these two kinds of centromere-positive MN were recorded.

Methods: CBMN assay in combination with fluorescent in situ hybridization using a human pancentromeric DNA probe were carried out on peripheral lymphocytes on 3 studies: (i) 10 untreated cancer patients vs 10 controls, (ii) 27 welders